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REACTIVITY IN SPACE MEDICINE

by V. V. Parin, P. V. Vasil'yev, and V. Ye. Belay

Paper presented at the XV International Astronautical Congress, Warsaw, September 7-12, 1964

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Translation of "K probleme reaktivnosti v kosmicheskoy meditsine"

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REACTIVITY IN SPACE MEDICINE

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Flights in modern airplanes and spacecraft are associated with noise, vibration, acceleration, and, in some cases, weightlessness. Considerable physical exertion and nervous and emotional stress are other adverse factors. Sometimes the magnitude and duration of these factors reach the limits of physiological endurance, at which point they can sharply reduce the pilot's physical efficiency and even impair his health, markedly changing the reactivity of his body.

Reactivity is the capacity of the body to respond in a certain way to environmental stimuli. It reflects, as it were, the state of the relationship (equilibrium) between the organism and the environment (Refs. 1, 2). As a result of environmental factors, reactivity may change considerably, sometimes assuming a pathological character. A. A. Bogomolets wrote: "Impairment of normal reactivity is the main factor in determining the possibility of disease arising, its course and outcome... It is essential to learn how to control the protective forces of the body, the best helper of the patient and physician."[1]

Hence, study of the influence of the environment (characteristics of nutrition, temperature conditions, barometric pressure, gaseous constituents of inhaled air, physical and neuropsychic factors, etc.) on the individual physiological reactivity of animals and man is of exceptional value. The concept of reactivity is inseparably bound up with resistance. The most important qualitative indicator of the body's reactivity is its resistance to injurious agents. But not every increase in reactivity is biologically desirable. For example, in anaphylactic shock reactivity increases, but at the expense of weakening resistance to various injuries. On the contrary, hibernation and hypothermia are known to entail a decrease in reactivity; but this decrease is an adaptive reaction that intensifies resistance to oxygen deficiency, anemia, accelerations, infections, and some other pathogenic agents (Refs. 15, 25, 31, 32, 41).

After having studied the nature of the changes in reactivity brought about by environmental factors, we are in a position to

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[1]Bogomolets, A. A., Izbrannyye trudy (Selected Works). Vol. 3, 295, 1958.

intelligently increase resistance to stresses, on the one hand, and to recommend appropriate therapeutic measures for any pathological conditions that may arise, on the other.

One of the major physical factors during flight is acceleration. Acceleration can easily be produced and accurately measured in the laboratory on a centrifuge. It has been extensively studied and there is a mass of experimental material on the body's reactions to it. The main pathogenetic elements in the resultant disorders have also been investigated.

Drugs have been widely used to induce directed and measured changes in function. By knowing the pharmacodynamics of a drug and its effects on different organs and systems as well as the pathogenesis of a process under study, the investigator is able to alter the body's reactivity and intensify its resistance to various factors. Naturally, not every drug, however theoretically justified, will produce the desired prophylactic or therapeutic effect because neither the pharmacodynamics of drugs nor the mechanisms of development of pathological processes are, in general, completely understood. Moreover, new elements in the pathogenesis of a particular disease and new properties of drugs are often manifested when new conditions affect the relationship between the organism and an experimental drug.

This article summarizes the experimental data obtained by the authors in collaboration with G. D. Glod, S. P. Kolchin, Ye. S. Sviridova, and S. V. Maslyanenko in their investigations of the relationship between reactivity and accelerations.

Our first objective was to determine whether resistance can be increased by changing function through the use of pharmacological agents. Attempts of this kind have already been made by Soviet (Refs. 3, 7, 8, 9, 12, 15, 16, 19, 27, 28, 29, 30) and foreign (Refs. 34, 35, 38, 39, 40) investigators. However, their results are conflicting, even in the case of the same drug. The reasons seem to be that they used different methods of producing accelerations, employed different criteria in evaluating effectiveness, and administered different doses of the drugs and at different times before the start of rotation. Incidentally, the latter two conditions largely determine the level of reactivity at the time of acceleration.

Our experimental animals were white mice, rats, rabbits, and dogs. Reactivity prior to acceleration was changed by using the following drugs: strychnine, epinephrine, norepinephrine, phenamine (Benzedrine), phenatine (product of condensation of phenamine and nicotinic acid), ephedrine, caffeine, Cardiazol, strophanthin-K, nitroglycerin, dibazol (2-benzylbenzimidazole hydrochloride), chloral hydrate, and pentothal sodium.

The main criterion used in evaluating the role of change in reactivity in affecting resistance to accelerations in the experiments on mice and rats was the survival rate, and in the experiments on rabbits and dogs--the length of time required for cardiac and respiratory functions to become impaired and the degree of impairment.

A comparative quantitative evaluation of the resistance of the experimental and control mice and rats was based on determination of the LD_{50} by Behrens' method (Ref. 33).

With optimum doses of drugs from different pharmacological groups--general and cardiovascular stimulants, anesthetics, etc.--and suitable times and methods of administration, we were able to favorably influence resistance to accelerations. The best results were obtained with strychnine, some sympathomimetics, and anesthetics. For example, in experiments on white mice injected five times with 0.1 mg/kg of strychnine once a day, the animals' resistance, as measured by the LD_{50}, was 6.6 units higher than that of the control (Figure 1). The survival rate following accelerations of 66 units was 21 percent higher than that of the control. The general condition of the mice before centrifugation was characterized by increased excitability, intensified tone of the striated muscles, and greater reactivity to acoustic and photic stimuli. Fewer injections (1-3) diminished the effectiveness of the drugs.

The favorable effect of strychnine was also reflected in the indicators of cardiac activity after accelerations in experiments on rabbits and dogs. For example, intact rabbits subjected to accelerations developed arrhythmia, extrasystole, and sinus bradycardia, which achieved critical values. One injection of 0.02 mg/kg of strychnine nitrate solution daily for three successive days reduced the degree of impairment in most cases and in two cases prevented impairment altogether (see table).

Dynamics of Pulse Rate in Rabbits Subjected to Accelerations in Control Experiments and After the Injection of Strychnine
(Mean Data)

Nature of experiments	No. of animals	Pulse rate before rotation	Pulse rate in relation to original value, percent								
			During rotation						After rotation		
Control	12	282	66	60.5	57	59	59.2	55.7	94.5	90.5	90.5
With injection of strychnine (0.02 mg/kg)	12	283	86.2	78	73	69	69.4	66.5	104	101	100.3

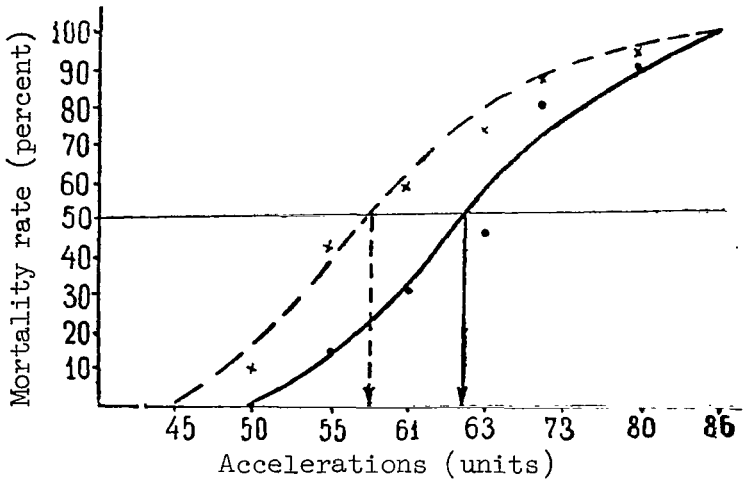

Figure 1. Curves of individual resistance of control and experimental (0.1 mg/kg of strychnine) mice to accelerations (LD_{50} determined by Behrens' method):

---------- control;
———————— experimental

The results were the same in the experiments on dogs.

Almost all of the drugs from the group of sympathomimetic amines, after determination of the optimum dose, proved to have a favorable effect in increasing the tolerance of lateral accelerations. In the experiments to determine the mean lethal value of the overload, it was found that the injection of 0.1 mg/kg of epidenphrine 30 minutes before rotation on a centrifuge increased resistance by 7.7 units, and 0.5 mg/kg of norepinephrine increased resistance by 3.3 units over the control (Figure 2).

The use of epinephrine in the experiments on rabbits reduced the degree of cardiovascular impairment, as shown by the less pronounced bradycardia (Figure 3).

The resistance of mice was likewise increased by the use of Benzedrine, especially in doses of 0.8-1 mg/kg. Either an increase or a decrease in the dose sharply reduced the drug's effectiveness, while a dose of 5 mg/kg had an adverse effect.

It is noteworthy that in the entire group of sympathomimetic amines tested, only phenatine did not have a beneficial effect. Being a product of the condensation of Benzedrine and nicotinic acid, this drug stimulates the central nervous system, whereas Benzedrine dilates the peripheral

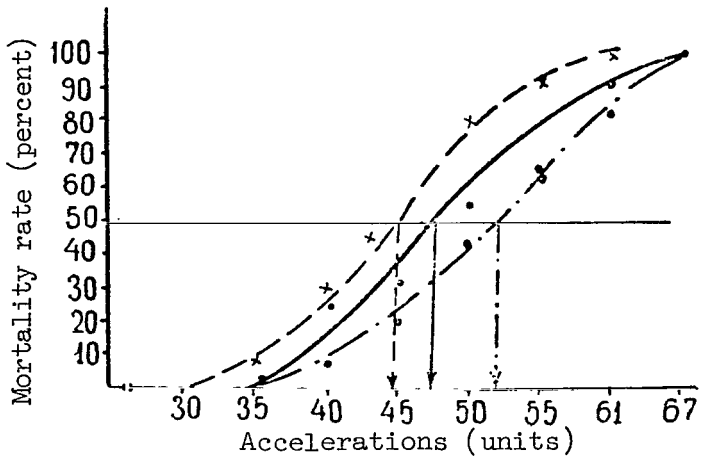

Figure 2. Curves of individual resistance to accelerations of control and experimental mice:

---------- control;
———————— with 0.5 mg/kg of norepinephrine;
.—.—.—.—.—.—. with 0.1 mg/kg of epinephrine

Figure 3. Effect of norepinephrine on change in the pulse rate of rabbits subjected to accelerations (9 units - 6 minutes)

▨ - control;
□ - with 0.2 mg/kg of norepinephrine

blood vessels, thus inducing a hypotensive effect. This aspect of its action was obviously a negative influence during accelerations.

A distinct increase in resistance to accelerations was noted after the administration of 0.5 mg/kg of ephedrine. Either a decrease or an increase in the dose sharply reduced its effectiveness.

The results of the experiments with adrenomimetics are consistent with the findings of some other investigators (Refs. 34, 40).

Of importance in the mechanism of the favorable action of phenylalkylamines during accelerations is that, in addition to a general hypertensive and tonic effect on the central nervous system, the volumetric flow rate of coronary blood increases and the oxygen supply of the myocardium improves (Ref. 18). It is particularly important to determine the optimum doses and times of administration of these drugs prior to rotation.

The significance of the original functional state of the organism resulting from administration of a drug is also apparent from the experiments with analeptics and anesthetics. For example, the survival rate of white mice injected with various doses of Cardiazol at the same time (30 minutes) before centrifugation differed. As shown in Figure 4, Cardiazol in doses ranging from 5 to 40 mg/kg caused a slight increase in resistance to accelerations, but an increase to 60 mg/kg resulted in a sharp decrease. The results were similar with chloral hydrate and pentothal sodium. A dose of 200 mg/kg of chloral hydrate 15 minutes before the start of rotation increased resistance by 2-10 units, whereas a dose of 400 mg/kg lowered it from 65 to 48 units (Figures 5 and 6).

A dose of 30 mg/kg of pentothal sodium increased the tolerance of accelerations by 4-19 units, while a dose of 100 mg/kg lowered it by 14 units. The negative results of experiments with large doses of chloral hydrate and pentothal sodium were probably due to a lowering of the functional level of the compensatory-adaptive mechanisms of the organism in response to unusually strong environmental stimuli. The findings were the same in many investigations of the effect produced by certain forms of oxygen deficiency (Refs. 14, 22, 24, 25, 26).

In summary, deliberate change in reactivity by means of pharmacological agents is a promising approach to the problem confronting aviation and space medicine of increasing human resistance to accelerations.

Another important aspect of reactivity is the sensitivity of the organism to pharmacological agents in the period following exposure to flight factors. It has been definitely established that on exposure to critical environmental factors sensitivity to drugs increases in some cases, decreases in others, and is distorted in still others. For example,

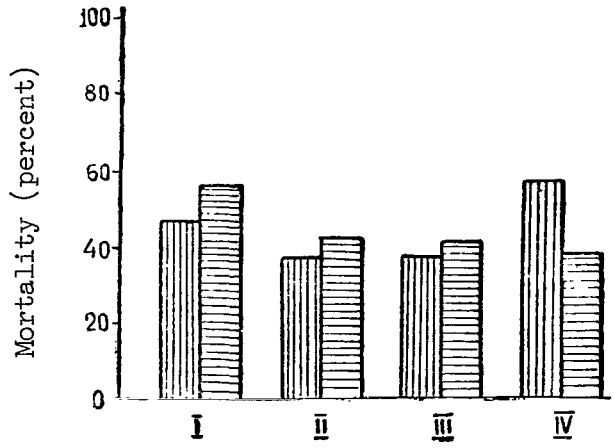

Figure 4. Effect of Cardiazol on the survival rate of control and experimental white mice
Doses of Cardiazol: I–5; II–20; III–40; IV–60 mg/kg

▨ - control;
▤ - experimental

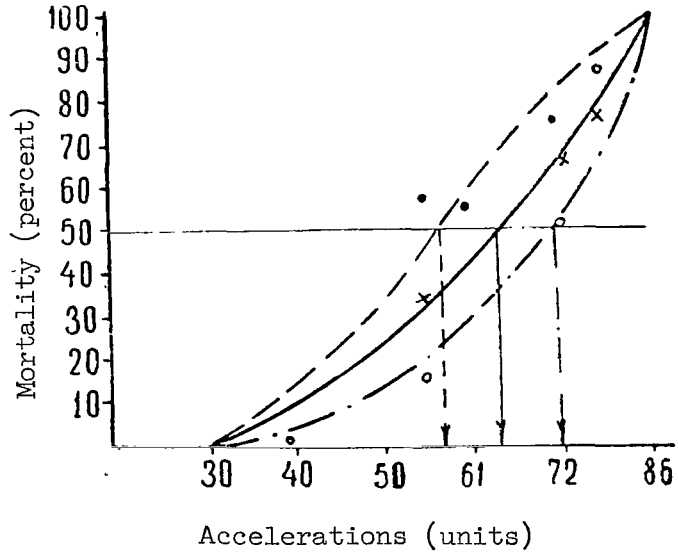

Figure 5. Curves of individual sensitivity of control and experimental white mice to accelerations:

---------- control;
———————— with 200 mg/kg of chloral hydrate;
—.—.—.—.—.— with 30 mg/kg of pentothal sodium

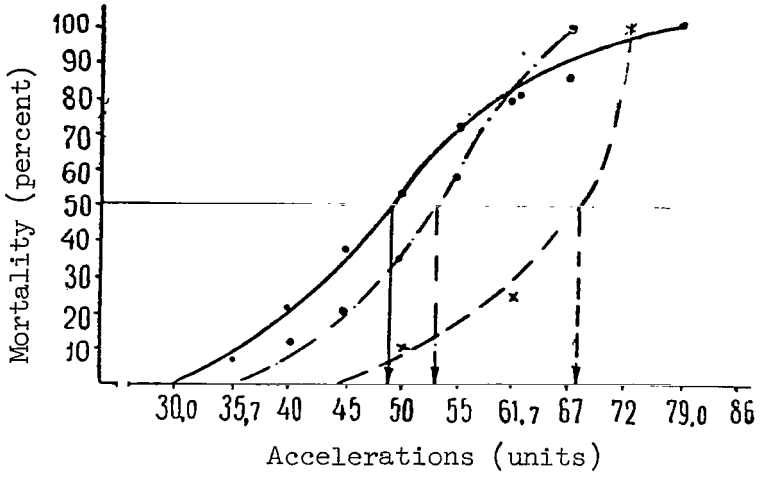

Figure 6. Curves of individual sensitivity of control and experimental white mice to accelerations:

---------- control;
———————— with 400 mg/kg of chloral hydrate;
—.—.—.—.—.— with 100 mg/kg of pentothal sodium

exposure to ionizing radiation caused marked changes in reactivity, depending on the time and severity of radiation sickness, to stimulants of hematopoiesis, systemic tonics, anesthetics, analeptics, vitamins, antihemorrhagic and diuretic agents, antibiotics, etc. (Refs. 5, 10, 20, 23).

After reviewing the literature, S. V. Anichkov (Ref. 4) concluded that environmental conditions significantly affect the action of pharmacological agents, even if they do not result in a pathological process.

We wish to emphasize the fact that the same drug used in the same dose, but with different functional state of the organism, may be a medicine or a poison (Refs. 4, 13, 21). Consequently, a knowledge of the action of pharmacological agents with reactivity of the organism altered by various environmental factors is absolutely essential, especially in connection with space flight, where the complex of factors present may cause marked changes in reactivity, where in some cases drugs can be taken without the participation of a physician, and where monitoring of the reactions to the drugs is difficult.

Our first experiments on animals (mice, rats, rabbits, dogs, and monkeys) yielded evidence of the need for more cautious administration of certain drugs after exposure to accelerations. The experiments on white mice and rats produced statistically significant data showing that comparatively slight accelerations (7 and 13 G's) affect their sensitivity

to anesthetics. The nature of the change is determined by the degree and duration of acceleration, and by the type of anesthetic used. For example, the duration of chloral hydrate anesthesia decreased after three minutes of accelerations of 13 G's, but after nine minutes it was double that of the control.

In the experiments with pentothal sodium, which acts chiefly on the subcortical divisions of the brain, the results were directly opposite. For example, after accelerations of the same intensity lasting three minutes, the duration of anesthesia increased 165 percent above the original value, whereas it dropped to 36.4 percent after nine minutes (Ref. 6).

The normal level of reactivity was restored only 1-1/2 to 2 hours after the ending of rotation. The results of these experiments show that temporary accelerations intensify the excitatory processes in the cerebral cortex (weakening of the effect of chloral hydrate), but by the law of negative induction strengthen the processes of inhibition (increase in sensitivity to pentothal sodium) in the subcortex. Prolonged accelerations, on the other hand, inhibit the cortical cells and disinhibit the subcortex.

The data on the effect of anesthetics undoubtedly require further study because their use in ordinary doses may be dangerous.

The results of experiments with cardiac glucosides provide convincing evidence that this group of drugs increases reactivity. For example, in experiments on rabbits, dogs and monkeys, it was found that strophanthin-K and convasid (aqueous solution of purified Convallaria majalis glucosides) in doses that are therapeutic for intact animals had a tonic effect in the period following exposure to intense accelerations (Figures 7 and 8). In most cases the EKG showed signs of marked impairment of conduction and myocardial excitability (extrasystole, heterogeneous rhythm, ciliary arrhythmia).

The depth of the changes varied from animal to animal, being determined by the degree of impairment of cardiac activity arising during rotation on a centrifuge and by the time elapsing since exposure. It will be noted that the drugs were always administered after complete normalization of all the EKG elements. Consequently, it is evident from the results of the experiments that normalization of the EKG after accelerations is not a reliable indicator of functional restoration of the myocardium.

The reaction to vasoconstrictors (epinephrine, norepinephrine) and vasodilators (nitroglycerin, papaverine) likewise changed. There was generally a deeper and longer increase (after epinephrine and norepinephrine) or decrease (after nitroglycerin and papaverine) in blood pressure.

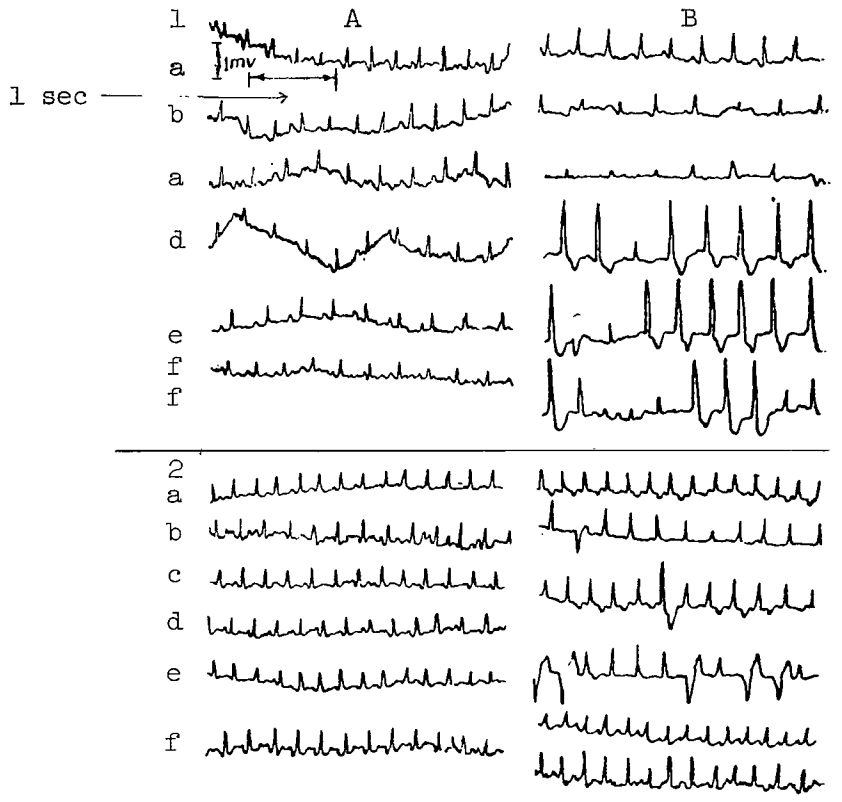

Figure 7. Changes in the EKG of the monkeys Khna (1) and Ineya (2) in response to the administration of 0.05 mg/kg of strophanthin-K:

A – before accelerations;
B – 20 minutes after accelerations;
a – EKG before administration of strophanthin;
b, c, d, e, f – 1, 3, 5, 7, and 15 minutes after administration of strophanthin

Thus, the results of our investigations indicate that accelerations change reactivity to various groups of pharmacological agents--anesthetics, cardiac glucosides, vasodilators, and vasoconstrictors.

These findings will naturally have to be taken into account in developing medical procedures for dealing with accelerations. They will throw more light on the mechanisms of the physiological reactions to accelerations because, as I. P. Pavlov wrote, "chemical substances are the most delicate analytical methods available to physiology."[1]

[1]Pavlov, I. P., Poln. sobr. soch. (Collected Writings), Vol. 2, Book I, 264, 1951.

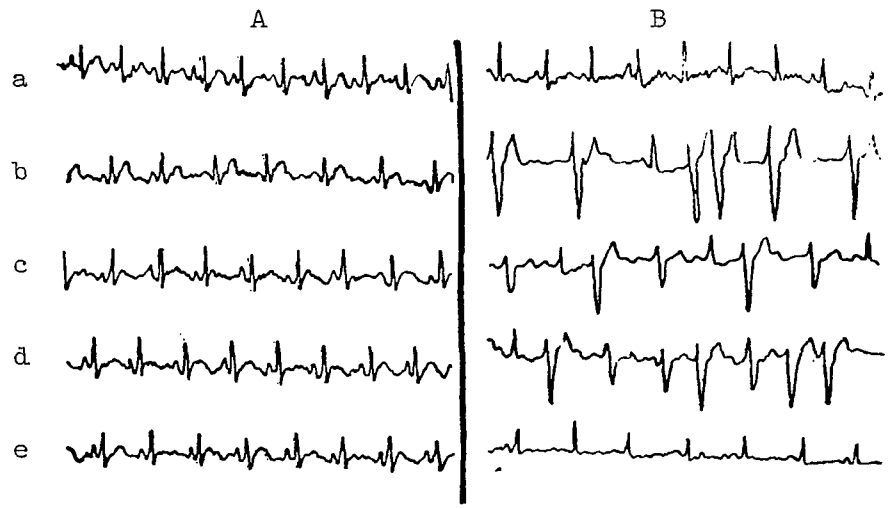

Figure 8. Changes in the EKG of a rabbit in response to the administration of convasid in a dose of 2 units per kg:

A – before accelerations;
B – 20 minutes after accelerations;
a – EKG before administration of convasid;
b, c, d, e – 1, 5, 7, and 10 minutes after administration of convasid

In conclusion, the results of our own observations and the experiments reported in the literature (Refs. 1, 2, 11, 17, 36, 37) indicate the need for broader study of the role played by reactivity in the effects of such factors as accelerations, vibrations, rolling motion, and weightlessness, and by the sensitivity of the organism to drugs from various pharmacological groups in the period following exposure to the above factors.

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